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# ACE gene I/D polymorphism in type 2 diabetes: the Gujarat population

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Table 1. Distribution of alleles and genotypes for the intron16 I/D polymorphism of ACE gene in people with and without type 2 diabetes

	N	Observed genotype counts (%)			Observed allele frequencies		Expected genotype counts <sup>a</sup>			
		1/1	I/D	D/D	1	D	1/1	I/D	D/D	$ ho ext{-}$ value*
No diabetes	445	139 (31.24)	199 (44.72)	107 (24.04)	0.536	0.464	127.72	221.17	95.68	0.102
Type 2 diabetic	290	76 (26.21)	108 (37.24)	106 (36.55)	0.448	0.552	58.29	143.55	88.45	0.0001
<i>p</i> -value Odds ratio		1.421	0.001 <sup>b</sup> 1.421 (1.152–1.754)		0.	001 <sup>c</sup>				

<sup>(95%</sup> CI)

**Key:** D = deletion; I = insertion.

We wish to draw attention to the increased frequency of ACE I/D (angiotensin converting enzyme insertion/deletion) polymorphisms in type 2 diabetic patients in the Gujurat region of India. The gene-encoding ACE is located on chromosome 17g23 and known to show I/D polymorphism of a 287 bp Alu-1-repetitive sequence (NCBI: AF118569; repeat region 14094-14381) in intron 16. This accounts for half of the variance of serum ACE levels in individuals homozygous for the insertion allele (II genotype) who have lower ACE levels than carriers of the deletion allele (ID and DD genotypes). Association studies of ACE I/D polymorphisms in people with type 2 diabetes of different

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ethnicities have yielded conflicting results,<sup>2</sup> so we undertook a case control study to investigate the association of ACE I/D polymorphism with type 2 diabetes in subjects from Gujarat.

Genomic DNA was isolated from whole blood of 290 (132 males and 158 females) type 2 diabetic patients (fasting blood glucose >180 mg/dl) and 445 (205 males and 240 females) ethnically matched non-diabetic individuals. The ACE I/D polymorphism was genotyped and scored by polymerase chain reaction (PCR).3

Significant differences in the genotype frequencies of I/I, I/D and D/D genotypes were observed between diabetic and nondiabetic subjects (p=0.001) suggesting an association of the ACE I/D polymorphism with type 2 diabetes. The allele frequencies for I and D alleles also differed significantly between diabetic and non-diabetic subjects (p=0.001). The type 2 diabetic population deviated from the Hardy-Weinberg equilibrium for the polymorphism (p=0.0001) and the non-diabetic population followed genetic equilibrium (p=0.102) (table 1).

A recent meta-analysis of ACE I/D polymorphism in type 2 diabetic showed significant association with the D allele in those of Caucasian and East Asian ethnicities.<sup>2</sup> Our study in the

<sup>&</sup>lt;sup>a</sup> Observed versus expected according to the Hardy-Weinberg equation.

<sup>&</sup>lt;sup>b</sup> No diabetes versus type 2 diabetes using the chi-square test with 3 × 2 contingency table.

 $<sup>^{\</sup>circ}$  No diabetes versus type 2 diabetes using the chi-square test with 2  $\times$  2 contingency table.

<sup>\*</sup>Values are significant at  $p \le 0.05$ .

populace of Gujarat (western India) together with others<sup>4,5</sup> suggests a strong link between ACE I/D polymorphism and type 2 diabetes at least in South Asian and Southwest Asian populations. Due to the metabolic and haemodynamic role of ACE. ACE I/D polymorphisms may be implicated in the pathogenesis of type 2 diabetic, especially in these Asian populations.

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